WEST Search History

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DATE: Sunday, July 23, 2006

Hide?	Set Name	Query	Hit Count
	DB=USPT; THES=ASSIGNEE; PLUR=YES; OP=ADJ		
☐	L8	L7	30
☐	L7	L6 and neoangiogenesis	30
☐	L6	L5	190
	DB=PGPB,USPT,EPAB,JPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ		
☐	L5	L3 and myocardium	633
☐	L4	L3 and revasculation	2
☐	L3	L2 and ischemic	2399
☐	L2	L1 and angiogenesis	6118
☐	L1	FGF	13852

END OF SEARCH HISTORY

- Search History will be lost after eight hours of inactivity.
- To combine searches use # before search number, e.g., #2 AND #6.
- Search numbers may not be continuous; all searches are represented.
- Click on query # to add to strategy

Search	Most Recent Queries	Time	Result
#8	Search **myocardium and neoangiogenesis and fgf**	11:24:53	11
#7	Search **myocardium and neoangeogenesis and FGF**	11:24:19	165
#6	Search **schumacher B and neoangeogenesis**	11:23:58	237
#2	Search **schumacher B 1998 and neoangeogenesis**	11:23:45	26
#1	Search **schumacher B 1998**	11:20:28	26

Clear History

```
=> FGF
L1        22523 FGF

=> myocardium
L2        87314 MYOCARDIUM

=> neoangiogenesis
L3         1190 NEOANGIOGENESIS

=> ischemic
L4       143748 ISCHEMIC

=> injection
L5       850510 INJECTION
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6642026 Stegmann T

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=> L1 and L4
L9          463 L1 AND L4

=> L9 and L2
L10          94 L9 AND L2

=> L3 and L1
L11          43 L3 AND L1

=> L2 and L11
L12           9 L2 AND L11

=> L5 and L10
L13          35 L5 AND L10

=> L5 and L11
L14           8 L5 AND L11

=> human and L13
L15          15 HUMAN AND L13
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L8 0 L6 AND L7

=> L1 and L4
L9 463 L1 AND L4

=> L9 and L2
L10 94 L9 AND L2

=> L3 and L1
L11 43 L3 AND L1

=> L2 and L11
L12 9 L2 AND L11

=> L5 and L10
L13 35 L5 AND L10

=> L5 and L11
L14 8 L5 AND L11

=> human and L13
L15 15 HUMAN AND L13

=> D L15 IBIB ABS 1-15

L15 ANSWER 1 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:612334 CAPLUS
DOCUMENT NUMBER: 143:126786
TITLE: Treatment of coronary or peripheral ischemia with heparin compound in combination with expression vector encoding angiogenic growth factor
INVENTOR(S): Uzan, Andre; Caron, Alexis
PATENT ASSIGNEE(S): Centelion, Fr.; Aventis Pharma S. A.
SOURCE: PCT Int. Appl., 41 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005063807	A2	20050714	WO 2004-EP14910	20041229
WO 2005063807	A3	20050929		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,

GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2003-532993P P 20031229

AB The present invention relates to novel methods of treating a patient having peripheral and/or coronary ischemic syndrome and also relates to a product comprising an expression vector encoding an angiogenic growth factor and a heparin compd., wherein the product is capable of acting in a synergistic manner to promote angiogenesis and arteriogenesis in skeletal and cardiac muscles. I.m. administration of plasmid NV1FGF, expressing human FGF-1, combined with s.c. administration of enoxaparin (low-mol.-wt. heparin) 14 days after induction of hindlimb ischemia greatly improved collateral vessel formation in the ischemic limb of hamsters when compared with saline-treated hamsters, enoxaparin, or NV1FGF gene therapy alone.

L15 ANSWER 2 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:521720 CAPLUS
DOCUMENT NUMBER: 143:20021
TITLE: Non-mitogenic FGF-2 protects against ischemia and/or reperfusion injury
INVENTOR(S): Kardami, Elissavet
PATENT ASSIGNEE(S): Can.
SOURCE: Can. Pat. Appl., 56 pp.
CODEN: CPXXEB
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CA 2390285	AA	20031211	CA 2002-2390285	20020611

PRIORITY APPLN. INFO.: CA 2002-2390285 20020611

AB Fibroblast growth factor-2 (FGF-2) is acutely cardioprotective towards non-ischemic as well as ischemic myocardium. The potent mitogenic activity of FGF- 2, however, may pose limitations to some of its clin. applications. In this study we examd. whether a recombinant FGF-2 mutant (S117A) that is no longer mitogenic retains cardioprotective properties.

Administration of S117A FGF-2 after 30 min of ischemia and during reperfusion of the ex vivo perfused rat heart resulted in significant protection (comparable to that of wild type FGF-2) against myocardial contractile dysfunction. In an in vivo study, rat myocardial infarction was induced by irreversible left coronary ligation; S117A FGF-2, or saline, were administered by direct intramyocardial injection into the ischemic left front ventricular wall. One day later: infarct size (assessed histol.), and plasma cTnT levels (assessed by Western blotting) were significantly decreased in the S117A FGF-2-, compared to the saline- treated control group, by 32.2% ($P<0.01$) or 28.5 % ($P<0.01$), resp.;systolic pressure, rates of contraction and relaxation and developed pressure, assessed in the Langendorff mode, were significantly increased in the S117A FGF-2 group compared to saline-treated group. One week after infarction, echocardiog. showed significantly improved contractile function (ejection fraction, fractional shortening), in the S117A FGF-2 or wild type FGF-2 treated hearts compared to the saline treated group. At 6 wk post infarction, however, S117 FGF -2-treated hearts had similar scar size and contractile function (systolic pressure, developed pressure, rates of contraction and relaxation, ejection fraction, fractional shortening) to the saline-treated group, while wild type FGF-2- treated hearts continued to display significantly improved contractility and reduced scar size. The wild type-, but not S117-, FGF-2-treated group had significantly increased microvessel d. at or near the scar area. It is concluded that acute cardioprotection by FGF-2 against ischemia and/or reperfusion -induced contractile dysfunction and tissue damage is independent of its mitogenic/ angiogenic activity. Thus the non-mitogenic, non-angiogenic S117 FGF-2 may serve as an agent of secondary injury prevention during early (one week) management of myocardial infarction and re-establishment of blood flow. Long-term protection of underperfused myocardium is likely to require increased vessel formation, and the mitogenic/angiogenic activity of wild type FGF-2.

L15 ANSWER 3 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:1074120 CAPLUS
DOCUMENT NUMBER: 142:32967
TITLE: Plasmid encoding fibroblast growth factor for the treatment of hypercholesterolemia or diabetes associated angiogenic defects
INVENTOR(S): Caron, Alexis; Emmanuel, Florence; Caron, Anne; Finiels, Francoise; Michelet, Sandrine; Schwartz, Bertrand; Rouy, Didier; Branellec, Didier
PATENT ASSIGNEE(S): Gencell S.a.S., Fr.
SOURCE: PCT Int. Appl., 88 pp.

CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004108167	A1	20041216	WO 2004-EP6903	20040604
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004244756	A1	20041216	AU 2004-244756	20040604
CA 2526792	AA	20041216	CA 2004-2526792	20040604
EP 1677831	A1	20060712	EP 2004-740314	20040604
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK				
US 2005096286	A1	20050505	US 2004-861906	20040607

PRIORITY APPLN. INFO.: US 2003-475959P P 20030605
US 2004-560915P P 20040409
US 2004-566193P P 20040428
WO 2004-EP6903 W 20040604

AB The present invention relates to the use of a plasmid encoding a fibroblast growth factor as therapeutic agent for the prevention and treatment of hypercholesterolemia or diabetes assocd. myocardial or skeletal angiogenic defects. The present invention also relates to a method for enhancing formation of both collateral blood vessels and arterioles in myocardial or skeletal ischemic tissues in a mammalian subject suffering from hypercholesterolemia or diabetes. The present invention further relates to a method of promoting collateral blood vessels in ischemic myocardial or skeletal tissues without inducing VEGF-A factor expression and causing edema in the treated muscles. In particular, NV1FGF (a human FGF-1 expression plasmid) is transferred to a rat model for hindlimb ischemia to access the potency of FGF-1 gene transfer of therapeutic angiogenesis in ischemic skeletal muscles. NV1FGF is shown to reverse the cholesterol-induced impairment of revascularization in a hamster model of hindlimb ischemia by promoting the growth of both

collateral vessels and arterioles in ischemic muscles exhibiting significantly decreased levels of gene expression compared with control muscles. Thus this study underscores the relevance of NV1FGF gene therapy to overcome perfusion defects in patients with PAD.

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 4 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:493523 CAPLUS
DOCUMENT NUMBER: 141:47325
TITLE: Method of producing biologically active human acidic fibroblast growth factor and its use in promoting angiogenesis
INVENTOR(S): Stegmann, Thomas J.; Kordyum, Vitaliy A.; Slavchenko, Iryna Yu.; Chernykh, Svitlana I.; Vozianov, Oleksandr F.
PATENT ASSIGNEE(S): Germany
SOURCE: U.S. Pat. Appl. Publ., 42 pp., Cont.-in-part of U.S. Ser. No. 929,945.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004115769	A1	20040617	US 2003-649480	20030827
US 2002122792	A1	20020905	US 1999-358780	19990722
US 2002155532	A1	20021024	US 2001-929945	20010815
US 6642026	B2	20031104		
US 2003054492	A1	20030320	US 2002-280864	20021024

PRIORITY APPLN. INFO.: US 1998-93962P P 19980724
US 1999-358780 A2 19990722
US 2000-225406P P 20000815
US 2001-929945 A2 20010815

AB The present invention relates to the treatment of coronary heart disease by revascularization therapy, and more particularly to the intramyocardial injection of a pharmaceutical compn. comprising a recombinant fibroblast growth factor-1 protein or a fragment of a recombinant fibroblast growth factor-1 protein, optionally, with a physiol. glue for inducing local neoangiogenesis in ischemic myocardium.
The invention also discloses methods of producing the recombinant fibroblast growth factor 1 protein and fragments. The methods involve phage-dependent delayed lysis of an Escherichia coli host cell for

high-level prodn of sol., recombinant protein. The effects of the human aFGF 154, 146, and 140 recombinant proteins on angiogenesis were compared to pure brain-derived aFGF using the model of new blood vessel formation in chicken embryo chorio-allantoic membrane. Induced neoangiogenesis was also found in the ischemic rat heart model. Recombinant FGF-1 was used clin. in combination with coronary artery bypass graft in patients with coronary heart disease and FGF-1140 was used as sole therapy in 20 patients with coronary heart disease.

L15 ANSWER 5 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:895952 CAPLUS
DOCUMENT NUMBER: 139:374668
TITLE: A randomized, double-blind, placebo-controlled trial of Ad5FGF-4 gene therapy and its effect on myocardial perfusion in patients with stable angina
AUTHOR(S): Grines, Cindy L.; Watkins, Matthew W.; Mahmarian, John J.; Iskandrian, Ami E.; Rade, Jeffrey J.; Marrott, Pran; Pratt, Craig; Kleiman, Neal
CORPORATE SOURCE: Angiogenic GENe Therapy (AGENT-2) Study Group, Department of Medicine, Section of Cardiology, William Beaumont Hospital, Royal Oak, MI, USA
SOURCE: Journal of the American College of Cardiology (2003), 42(8), 1339-1347
CODEN: JACCDI; ISSN: 0735-1097
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The primary objective of this study was to det. whether intracoronary administration of the adenoviral gene for fibroblast growth factor (Ad5FGF-4) can improve myocardial perfusion compared with placebo. Animal studies and observational clin. studies have shown improvement in perfusion of the ischemic myocardium using genes encoding angiogenic growth factors; however, randomized, double-blind data in humans are lacking. We performed a randomized, double-blind, placebo-controlled trial of intracoronary injection of 1010 adenoviral particles contg. a gene encoding fibroblast growth factor (Ad5FGF-4) to det. the effect on myocardial perfusion. Fifty-two patients with stable angina and reversible ischemia comprising >9% of the left ventricle on adenosine single-photon emission computed tomog. (SPECT) imaging were randomized to gene therapy (n = 35) or placebo (n = 17). Clin. follow-up was performed, and 51 (98%) patients underwent a second adenosine SPECT scan after 8 wk. Overall (n = 52), the mean total perfusion defect size at baseline was 32.4% of the left ventricle, with 20% reversible ischemia and 12.5% scar. At eight weeks, Ad5FGF-4 injection resulted in a significant redn. of ischemic

defect size (4.2% abs., 21% relative; $p < 0.001$) and placebo-treated patients had no improvement ($p = 0.32$). Although the change in reversible perfusion defect size between Ad5FGF-4 and placebo was not significant (4.2% vs. 1.6%, $p = 0.14$), when a single outlier was excluded a significant difference was obsd. (4.2% vs. 0.8%, $p < 0.05$). Ad5FGF-4 was well tolerated and did not result in any permanent adverse sequelae. Intracoronary injection of Ad5FGF-4 showed an encouraging trend for improved myocardial perfusion; however, further studies of therapeutic angiogenesis with Ad5FGF-4 will be necessary.

REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 6 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:610064 CAPLUS
DOCUMENT NUMBER: 139:160389
TITLE: Techniques and compositions for treating cardiovascular disease by in vivo gene delivery of angiogenic peptides and proteins
INVENTOR(S): Hammond, H. Kirk; Dillmann, Wolfgang; Giordano, Frank J.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 51 pp., Cont.-in-part of U.S. Ser. No. 609,080, abandoned.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 12
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003148968	A1	20030807	US 2001-847936	20010503
US 5792453	A	19980811	US 1995-485472	19950607
US 6100242	A	20000808	US 1997-722271	19971229
US 6174871	B1	20010116	US 1998-132167	19980810
WO 9940945	A2	19990819	WO 1999-US2702	19990209
WO 9940945	A3	19990930		

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 9947541 A1 19991125 AU 1999-47541 19990910
WO 2001034208 A1 20010517 WO 2000-US30345 20001103
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
ZA 2002003303 A 20030526 ZA 2002-3303 20020425
WO 2002089856 A1 20021114 WO 2002-US13990 20020503
WO 2002089856 C1 20040401
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
AU 2002305346 A1 20021118 AU 2002-305346 20020503
US 2004132190 A1 20040708 US 2003-741907 20031219
PRIORITY APPLN. INFO.: US 1995-396207 B2 19950228
US 1995-485472 A2 19950607
US 1997-852779 B1 19970506
US 1997-722271 A2 19971229
US 1998-21773 B2 19980211
US 1998-68102 B2 19980430
US 1998-132167 A1 19980810
WO 1999-US2702 A2 19990209
US 1999-435156 B2 19991105
US 2000-609080 B2 20000630
WO 2000-US30345 A2 20001103
US 1995-481122 B2 19950607
AU 1996-50287 A3 19960227
WO 1996-US2631 W 19960227
US 1996-660387 B1 19960607
US 1998-98174 B1 19980616
US 2000-664127 A3 20000918
US 2001-847936 A 20010503
WO 2002-US13990 W 20020503

AB Methods are provided for treating patients with cardiovascular disease,

including heart disease and peripheral vascular disease. The preferred methods of the present invention involve in vivo delivery of genes, encoding angiogenic proteins or peptides, to the myocardium or to peripheral ischemic tissue, by introduction of a vector contg. the gene into a blood vessel supplying the heart or into a peripheral ischemic tissue. A kit comprising a gene therapy compn., a device for introducing the compn. into a blood vessel or tissue in vivo, and a vasoactive agent is also claimed.

L15 ANSWER 7 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:674561 CAPLUS
DOCUMENT NUMBER: 137:196045
TITLE: Induction of neoangiogenesis in ischemic myocardium using a pharmaceutical composition containing FGF-1 and a physiological glue
INVENTOR(S): Stegmann, Thomas J.
PATENT ASSIGNEE(S): Germany
SOURCE: U.S. Pat. Appl. Publ., 16 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002122792	A1	20020905	US 1999-358780	19990722
US 2004115769	A1	20040617	US 2003-649480	20030827

PRIORITY APPLN. INFO.: US 1998-93962P P 19980724
US 1999-358780 A2 19990722
US 2000-225406P P 20000815
US 2001-929945 A2 20010815

AB The present invention relates to the treatment of coronary heart disease by revascularization therapy, and more particularly to the intramyocardial injection of a pharmaceutical compn. comprising fibroblast growth factor-1 and a physiol. glue for inducing local neoangiogenesis in ischemic myocardium.

L15 ANSWER 8 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:359425 CAPLUS
DOCUMENT NUMBER: 131:14116
TITLE: New approaches to coronary heart disease: induction of neovascularization by growth factors
AUTHOR(S): Stegmann, Thomas J.
CORPORATE SOURCE: Department of Thoracic and Cardiovascular Surgery, Fulda Medical Center, Fulda, Germany

SOURCE: BioDrugs (1999), 11(5), 301-308
CODEN: BIDRF4; ISSN: 1173-8804
PUBLISHER: Adis International Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 51 refs. Currently available approaches for treating human coronary heart disease aim to relieve symptoms and the risk of myocardial infarction by reducing myocardial oxygen demand (drugs), preventing further disease progression (drugs), restoring coronary blood flow either pharmacol. (thrombolysis) or mech. (angioplasty), or bypassing the stenotic lesions and obstructed coronary artery segments (surgery). Direct gene therapy, as well as gene-derived therapy, esp. by angiogenic growth factors, is emerging as a potential new treatment for cardiovascular disease. After extensive exptl. research on angiogenic growth factors, the first clin. studies on patients with coronary heart disease or peripheral vascular lesions are being performed. The polypeptides fibroblast growth factor (FGF) and vascular endothelial growth factor seem to be effective in initiating neovascularization (neo-angiogenesis) in hypoxic or ischemic tissues. The first clin. study on patients with coronary heart disease treated by local injection of FGF-1 into the compromised underperfused myocardial tissue showed a 3-fold increase of capillary d. mediated by the growth factor. Angiogenic therapy of the human myocardium introduces a new modality of treatment for coronary heart disease in terms of regulation of blood vessel growth. Beyond drug therapy, angioplasty and bypass surgery, this therapy may evolve to be a fourth principle of treatment of atherosclerotic cardiovascular disease.
REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 9 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:798674 CAPLUS
DOCUMENT NUMBER: 130:119673
TITLE: FGF-1: a human growth factor in the induction of neoangiogenesis
AUTHOR(S): Stegmann, Thomas J.
CORPORATE SOURCE: Department of Thoracic and Cardiovascular Surgery, Fulda Medical Center, Fulda, Germany
SOURCE: Expert Opinion on Investigational Drugs (1998), 7(12), 2011-2015
CODEN: EOIDER; ISSN: 1354-3784
PUBLISHER: Ashley Publications
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 25 refs. Currently available approaches for treating human coronary heart disease aim to relieve symptoms and the risk of myocardial infarction either by reducing myocardial oxygen demand, preventing further disease progression, restoring coronary blood flow pharmacol. or mech., or bypassing the stenotic lesions and obstructed coronary artery segments. Gene therapy, esp. using angiogenic growth factors, has emerged recently as a potential new treatment for cardiovascular disease. Following extensive exptl. research on angiogenic growth factors, the first clin. studies on patients with coronary heart disease and peripheral vascular lesions have been performed. The polypeptides fibroblast growth factor (FGF) and vascular endothelial growth factor (VEGF) appear to be particularly effective in initiating neovascularization (neo-angiogenesis) in hypoxic or ischemic tissues. The first clin. study on patients with coronary heart disease treated by local intramyocardial injection of FGF-1 showed a 3-fold increase of capillary d. mediated by the growth factor. Angiogenic therapy of the human myocardium introduces a new modality of treatment for coronary heart disease in terms of regulation of blood vessel growth. Beyond drug therapy, angioplasty and bypass surgery, this new approach may evolve into a fourth principle of treatment of atherosclerotic cardiovascular disease.
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 10 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:153567 CAPLUS
DOCUMENT NUMBER: 128:239666
TITLE: Induction of neoangiogenesis in ischemic myocardium by human growth factors: first clinical results of a new treatment of coronary heart disease
AUTHOR(S): Schumacher, B.; Pecher, P.; Von Specht, B. U.; Stegmann, Th.
CORPORATE SOURCE: Klinik fur Thorax-, Herz und Gefasschirurgie, Klinikum Fulda, Fulda, D-36043, Germany
SOURCE: Circulation (1998), 97(7), 645-650
CODEN: CIRCAZ; ISSN: 0009-7322
PUBLISHER: Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The present article is a report of our animal expts. and also of the first clin. results of a new treatment for coronary heart disease using the human growth factor FGF-I (basic fibroblast growth factor) to induce neoangiogenesis in the ischemic myocardium. FGF-I was obtained from strains of

Escherichia coli by genetic engineering, then isolated and highly purified. Several series of animal expts. demonstrated the apathogenic action and neoangiogenic potency of this factor. After successful conclusion of the animal expts., it was used clin. for the first time. FGF-I (0.01 mg/kg body wt.) was injected close to the vessels after the completion of internal mammary artery (IMA)/left anterior descending coronary artery (LAD) anastomosis in 20 patients with three-vessel coronary disease. All the patients had addnl. peripheral stenoses of the LAD or one of its diagonal branches. Twelve weeks later, the IMA bypasses were selectively imaged by intra-arterial digital subtraction angiog. and quant. evaluated. In all the animal expts., the development of new vessels in the ischemic myocardium could be demonstrated angiog. The formation of capillaries could also be demonstrated in humans and was found in all cases around the site of injection. A capillary network sprouting from the proximal part of the coronary artery could be shown to have bypassed the stenoses and rejoined the distal parts of the vessel. We believe that the use of FGF-I for myocardial revascularization is in principle a new concept and that it may be particularly suitable for patients with addnl. peripheral stenoses that cannot be revascularized surgically.

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 11 OF 15 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN

ACCESSION NUMBER: 2005:525488 BIOSIS
DOCUMENT NUMBER: PREV200510315451
TITLE: Intramyocardial injection of fibroblast growth factor-1 for treatment of refractory angina pectoris: The initial US experience.
AUTHOR(S): Wagoner, Lynne E. [Reprint Author]; Snavely, Daniel D.; Conway, Ginger A.; Hauntz, Elizabeth A.; Merrill, Walter H.
CORPORATE SOURCE: Univ Cincinnati, Cincinnati, OH USA
SOURCE: Circulation, (OCT 26 2004) Vol. 110, No. 17, Suppl. S, pp. 395.
Meeting Info.: 77th Scientific Meeting of the American-Heart-Association. New Orleans, LA, USA. November 07 -10, 2004. Amer Heart Assoc.
CODEN: CIRCAZ. ISSN: 0009-7322.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Dec 2005
Last Updated on STN: 1 Dec 2005

AB Coronary heart disease is the leading cause of death in the US. Despite advancing technology, a significant number of patients have refractory angina not amenable to revascularization resulting in a negative impact on quality of life. Fibroblast growth factors (FGF) have been shown to induce angiogenesis in ischemic territories. The only study of the efficacy of intramyocardial injection of FGF-1 was done in Europe by Stegmann et al. Methods: The Phase 1, open label, dose escalation, multicenter trial to evaluate safety, and efficacy of human FGF-1 for refractory angina pectoris is underway in the US. Eligible subjects were not candidates for revascularization and had a 3 month history of refractory CCS Angina Class III to IV with reversible myocardial ischemia. At baseline subjects underwent physical and fundoscopic exam, CCS Angina scores and Seattle Angina Questionnaires (SAQ). Myocardial SPECT-thallium, rest/stress echocardiograms, and coronary angiography with left ventriculography were performed. The intramyocardial FGF-1 was administered via mini-thoracotomy. Each patient received two injections of 1 g/kg. Testing was repeated after 12 weeks. Results: At 12 weeks there was evidence of increased area of contrast distribution and myocardial blush via angiography and increased perfusion via SPECT in the treatment areas. CCS Angina Class improved (Figure 1) as did SAQ scores. No major adverse events, malignancies, or ophthalmologic changes were seen. Conclusion: Intramyocardial injection of FGF-1 appears to be safe. The early results are promising. We await the results of the entire 32 patient cohort.[GRAPHICS]

L15 ANSWER 12 OF 15 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2005:30520 BIOSIS
DOCUMENT NUMBER: PREV200500033062
TITLE: Angiogenesis: A new therapeutic horizon in patients with stable angina pectoris.
Original Title: La angiogenesis, nuevo horizonte terapeutico en pacientes con angina cronica estable..
AUTHOR(S): Mautner, Branco [Reprint Author]; Cabello, Mariana; Garrido, Sol
CORPORATE SOURCE: Fdn Favaloro, ICYCC, Av Belgrano 1746, RA-1093, Buenos Aires, DF, Argentina
SOURCE: Prensa Medica Argentina, (August 2004) Vol. 91, No. 6, pp. 444-446. print.
CODEN: PMARAU. ISSN: 0032-745X.
DOCUMENT TYPE: Article
LANGUAGE: Spanish
ENTRY DATE: Entered STN: 12 Jan 2005
Last Updated on STN: 12 Jan 2005

AB Ischemic heart disease results from lack of oxygen and delayed removal of metabolites because of inadequate perfusion. The term ischemic heart disease refers to an abnormality of the heart in which there is Ischemia of the myocardium. One of the clinical manifestations of ischemic heart disease is stable chronic angina. Angina consists of chest pain that is episodic, usually brief, and caused by transient myocardial ischemia. Angina is the most common symptom of ischemic heart disease and requires immediate attention as to the diagnosis and extent of disease. At present, the therapeutic tools available for the treatment of ischemic heart disease are wide and not only related with medical therapy but also include two main surgical techniques: transluminal angioplasty and direct revascularization. Surgical management is recommended for patients initially assigned to medical therapy without improvement in their symptomatology. For this population also recently have emerged different therapeutic approaches not conventional in order to improve the quality of life and to prevent the apparition of coronary events. Angiogenic gene therapy appears to be a new valid option for the treatment of coronary artery disease, considering the intracoronary injection of genic substances in patients with chronic stable angina. The authors present a pilot study with a protocol for angiogenic gene therapy with adenovirus-5 with special reference to intracoronary recombinant human vascular endothelial growth myocardial perfusion of Ad 5 F GF-4 gene, and its effect in patients with angina. Initially encouraged by evidence, further studies are required to arrive to definitive conclusions. These considerations are commented in the article.

L15 ANSWER 13 OF 15 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2003:547302 BIOSIS
DOCUMENT NUMBER: PREV200300549128
TITLE: A randomized, double-blind, placebo-controlled trial of Ad5FGF-4 gene therapy and its effect on myocardial perfusion in patients with stable angina.
AUTHOR(S): Grines, Cindy L. [Reprint Author]; Watkins, Matthew W.; Mahmarian, John J.; Iskandrian, Ami E.; Rade, Jeffrey J.; Marrott, Pran; Pratt, Craig; Kleiman, Neal
CORPORATE SOURCE: Division of Cardiology, William Beaumont Hospital, 3601 West 13 Mile Road, 3rd Floor Heart Center, Royal Oak, MI, 48073-6769, USA
cgrines@beaumonthospitals.com
SOURCE: Journal of the American College of Cardiology, (October 15 2003) Vol. 42, No. 8, pp. 1339-1347. print.
ISSN: 0735-1097 (ISSN print).
DOCUMENT TYPE: Article

LANGUAGE: English
ENTRY DATE: Entered STN: 19 Nov 2003
Last Updated on STN: 19 Nov 2003

AB OBJECTIVES The primary objective of this study was to determine whether intracoronary administration of the adenoviral gene for fibroblast growth factor (Ad5FGF-4) can improve myocardial perfusion compared with placebo. BACKGROUND Animal studies and observational clinical studies have shown improvement in perfusion of the ischemic myocardium using genes encoding angiogenic growth factors; however, randomized, double-blind data in humans are lacking. METHODS We performed a randomized, double-blind, placebo-controlled trial of intracoronary injection of 1010 adenoviral particles containing a gene encoding fibroblast growth factor (Ad5FGF-4) to determine the effect on myocardial perfusion. Fifty-two patients with stable angina and reversible ischemia comprising >9% of the left ventricle on adenosine single-photon emission computed tomography (SPECT) imaging were randomized to gene therapy (n = 35) or placebo (n = 17). Clinical follow-up was performed, and 51 (98%) patients underwent a second adenosine SPECT scan after 8 weeks. RESULTS Overall (n = 52), the mean total perfusion defect size at baseline was 32.4% of the left ventricle, with 20% reversible ischemia and 12.5% scar. At eight weeks, Ad5FGF-4 injection resulted in a significant reduction of ischemic defect size (4.2% absolute, 21% relative; $p < 0.001$) and placebo-treated patients had no improvement ($p = 0.32$). Although the change in reversible perfusion defect size between Ad5FGF-4 and placebo was not significant (4.2% vs. 1.6%, $p = 0.14$), when a single outlier was excluded a significant difference was observed (4.2% vs. 0.8%, $p < 0.05$). Ad5FGF-4 was well tolerated and did not result in any permanent adverse sequelae. CONCLUSIONS Intracoronary injection of Ad5FGF-4 showed an encouraging trend for improved myocardial perfusion; however, further studies of therapeutic angiogenesis with Ad5FGF-4 will be necessary.

L15 ANSWER 14 OF 15 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2000:517402 BIOSIS
DOCUMENT NUMBER: PREV200000517402
TITLE: Induction of myocardial neoangiogenesis by human growth factors. A new therapeutic option in coronary heart disease.
AUTHOR(S): Stegmann, Thomas J. [Reprint author]; Hoppert, Thomas; Schneider, Andre; Gemeinhardt, Stefan; Koecher, Michael; Ibing, Rainer; Strupp, Gerhard
CORPORATE SOURCE: Klinik fuer Thorax-, Herz- und Gefaesschirurgie, Klinikum Fulda, Pacelliallee 4, D-36043, Fulda, Germany
SOURCE: Herz, (September, 2000) Vol. 25, No. 6, pp. 589-599. print.

ISSN: 0340-9937.
DOCUMENT TYPE: Article
LANGUAGE: German
ENTRY DATE: Entered STN: 29 Nov 2000
Last Updated on STN: 11 Jan 2002

AB Currently available approaches for treating human coronary heart disease aim to relieve symptoms and the risk of myocardial infarction either by reducing myocardial oxygen demand, preventing further disease progression, restoring coronary blood flow pharmacologically or mechanically, or bypassing the stenotic lesions and obstructed coronary artery segments. Gene therapy, especially using angiogenic growth factors, has emerged recently as a potential new treatment for cardiovascular disease. Following extensive experimental research on angiogenic growth factors, the first clinical studies on patients with coronary heart disease and peripheral vascular lesions have been performed. The polypeptides fibroblast growth factor (FGF) and vascular endothelial growth factor (VEGF) appear to be particularly effective in initiating neovascularization (neoangiogenesis) in hypoxic or ischemic tissues. The first clinical study on patients with coronary heart disease treated by local intramyocardial injection of FGF-1 showed a 3-fold increase of capillary density mediated by the growth factor. Also, angiogenic growth factor injection intramyocardially as sole therapy for end-stage coronary disease showed an improvement of myocardial perfusion in the target areas as well as a reduction of symptoms and an increase in working capacity. Angiogenic therapy of the human myocardium introduces a new modality of treatment for coronary heart disease in terms of regulation of blood vessel growth. Beyond drug therapy, angioplasty and bypass surgery, this new approach may evolve into a fourth principle of treatment of atherosclerotic cardiovascular disease.

L15 ANSWER 15 OF 15 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1998:131418 BIOSIS
DOCUMENT NUMBER: PREV199800131418
TITLE: Induction of neoangiogenesis in ischemic myocardium by human growth factors: First clinical results of a new treatment of coronary heart disease.
AUTHOR(S): Schumacher, B. [Reprint author]; Pecher, P.; Von Specht, B. U.; Stegmann, T.
CORPORATE SOURCE: Klinik fuer Thorax-, Herz und Gefasschirurgie, Klinikum Fulda, Pacelliallee 4, D-36043 Fulda, Germany
SOURCE: Circulation, (Feb. 24, 1998) Vol. 97, No. 7, pp. 645-650. print.

CODEN: CIRCAZ. ISSN: 0009-7322.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 20 Mar 1998
Last Updated on STN: 20 Mar 1998
AB Background-The present article is a report of our animal experiments and also of the first clinical results of a new treatment for coronary heart disease using the human growth factor FGF-I (basic fibroblast growth factor) to induce neoangiogenesis in the ischemic myocardium. Methods and Results-FGF -I was obtained from strains of Escherichia coli by genetic engineering, then isolated and highly purified. Several series of animal experiments demonstrated the apadiogenic action and neoangiogenic potency of this factor. After successful conclusion of the animal experiments, it was used clinically for the first time. FGF-I (0.01 mg/kg body weight) was injected close to the vessels after the completion of internal mammary artery (IMA)/left anterior descending coronary artery (LAD) anastomosis in 20 patients with three-vessel coronary disease. AU the patients had additional peripheral stenoses of the LAD or one of its diagonal branches. Twelve weeks later, the IMA bypasses were selectively imaged by intra-arterial digital subtraction angiography and quantitatively evaluated. In all the animal experiments, the development of new vessels in the ischemic myocardium could be demonstrated angiographically. The formation of capillaries could also be demonstrated in humans and was found in all cases around the site of injection. A capillary network sprouting from the proximal part of the coronary artery could be shown to have bypassed the stenoses and rejoined the distal parts of the vessel. Conclusions-We believe that the use of FGF-I for myocardial revascularization is in principle a new concept and that it may be particularly suitable for patients with additional peripheral stenoses that cannot be revascularized surgically.

=> L14 IBIB ABS 1-8
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L14 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:493523 CAPLUS
DOCUMENT NUMBER: 141:47325
TITLE: Method of producing biologically active human acidic fibroblast growth factor and its use in promoting angiogenesis
INVENTOR(S): Stegmann, Thomas J.; Kordyum, Vitaliy A.; Slavchenko, Iryna Yu.; Chernykh, Svitlana I.; Vozianov, Oleksandr F.
PATENT ASSIGNEE(S): Germany
SOURCE: U.S. Pat. Appl. Publ., 42 pp., Cont.-in-part of U.S. Ser. No. 929,945.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004115769	A1	20040617	US 2003-649480	20030827
US 2002122792	A1	20020905	US 1999-358780	19990722
US 2002155532	A1	20021024	US 2001-929945	20010815
US 6642026	B2	20031104		
US 2003054492	A1	20030320	US 2002-280864	20021024

PRIORITY APPLN. INFO.: US 1998-93962P P 19980724
US 1999-358780 A2 19990722
US 2000-225406P P 20000815
US 2001-929945 A2 20010815

L14 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:674561 CAPLUS
DOCUMENT NUMBER: 137:196045
TITLE: Induction of neoangiogenesis in ischemic myocardium using a pharmaceutical composition containing FGF-1 and a physiological glue
INVENTOR(S): Stegmann, Thomas J.
PATENT ASSIGNEE(S): Germany
SOURCE: U.S. Pat. Appl. Publ., 16 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002122792	A1	20020905	US 1999-358780	19990722
US 2004115769	A1	20040617	US 2003-649480	20030827

PRIORITY APPLN. INFO.: US 1998-93962P P 19980724
US 1999-358780 A2 19990722
US 2000-225406P P 20000815
US 2001-929945 A2 20010815

L14 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:873298 CAPLUS
DOCUMENT NUMBER: 134:37052
TITLE: Sulfated glucosamine glycans as VEGF and neoangiogenesis inhibitors
INVENTOR(S): Ishihara, Masayuki; Ono, Katsuaki; Suzuki, Kiyoshi
PATENT ASSIGNEE(S): Seikagaku Kogyo Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 14 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2000344674	A2	20001212	JP 2000-99433	20000331

PRIORITY APPLN. INFO.: JP 1999-93472 A 19990331

L14 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:798674 CAPLUS
DOCUMENT NUMBER: 130:119673
TITLE: FGF-1: a human growth factor in the induction of neoangiogenesis
AUTHOR(S): Stegmann, Thomas J.
CORPORATE SOURCE: Department of Thoracic and Cardiovascular Surgery, Fulda Medical Center, Fulda, Germany
SOURCE: Expert Opinion on Investigational Drugs (1998), 7(12), 2011-2015
CODEN: EOIDER; ISSN: 1354-3784
PUBLISHER: Ashley Publications
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:153567 CAPLUS
DOCUMENT NUMBER: 128:239666
TITLE: Induction of neoangiogenesis in ischemic myocardium by human growth factors: first clinical results of a new treatment of coronary heart disease
AUTHOR(S): Schumacher, B.; Pecher, P.; Von Specht, B. U.; Stegmann, Th.
CORPORATE SOURCE: Klinik fur Thorax-, Herz und Gefasschirurgie, Klinikum Fulda, Fulda, D-36043, Germany
SOURCE: Circulation (1998), 97(7), 645-650
CODEN: CIRCAZ; ISSN: 0009-7322
PUBLISHER: Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 6 OF 8 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2000:517402 BIOSIS
DOCUMENT NUMBER: PREV200000517402
TITLE: Induction of myocardial neoangiogenesis by human growth factors. A new therapeutic option in coronary heart disease.
AUTHOR(S): Stegmann, Thomas J. [Reprint author]; Hoppert, Thomas; Schneider, Andre; Gemeinhardt, Stefan; Koecher, Michael; Ibing, Rainer; Strupp, Gerhard
CORPORATE SOURCE: Klinik fuer Thorax-, Herz- und Gefaesschirurgie, Klinikum Fulda, Pacelliallee 4, D-36043, Fulda, Germany
SOURCE: Herz, (September, 2000) Vol. 25, No. 6, pp. 589-599. print.
ISSN: 0340-9937.
DOCUMENT TYPE: Article
LANGUAGE: German
ENTRY DATE: Entered STN: 29 Nov 2000
Last Updated on STN: 11 Jan 2002

L14 ANSWER 7 OF 8 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1998:131418 BIOSIS
DOCUMENT NUMBER: PREV199800131418
TITLE: Induction of neoangiogenesis in ischemic myocardium by human growth factors: First clinical results of a new treatment of coronary heart disease.
AUTHOR(S): Schumacher, B. [Reprint author]; Pecher, P.; Von Specht, B.

U.; Stegmann, T.
CORPORATE SOURCE: Klinik fuer Thorax-, Herz und Gefasschirurgie, Klinikum Fulda, Pacelliallee 4, D-36043 Fulda, Germany
SOURCE: Circulation, (Feb. 24, 1998) Vol. 97, No. 7, pp. 645-650. print.
CODEN: CIRCAZ. ISSN: 0009-7322.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 20 Mar 1998
Last Updated on STN: 20 Mar 1998

L14 ANSWER 8 OF 8 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1991:504041 BIOSIS
DOCUMENT NUMBER: PREV199192127001; BA92:127001
TITLE: TUMOR GROWTH DEPENDENT ON KAPOSI'S SARCOMA-DERIVED
FIBROBLAST GROWTH FACTOR INHIBITED BY PENTOSAN POLYSULFATE.
AUTHOR(S): WELLSTEIN A [Reprint author]; ZUGMAIER G; CALIFANO J A III;
KERN F; PAIK S; LIPPMAN M E
CORPORATE SOURCE: VINCENT T LOMBARDI CANCER RES CENTER, GEORGETOWN UNIV MED
CENTER, 3800 RESERVOIR ROAD NW, WASHINGTON, DC 20007, USA
SOURCE: Journal of the National Cancer Institute (Bethesda), (1991) Vol. 83, No. 10, pp. 716-720.
CODEN: JNCIEQ. ISSN: 0027-8874.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 12 Nov 1991
Last Updated on STN: 12 Nov 1991

=> D L12 IBIB ABS 1-9

L12 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:493523 CAPLUS
DOCUMENT NUMBER: 141:47325
TITLE: Method of producing biologically active human acidic fibroblast growth factor and its use in promoting angiogenesis
INVENTOR(S): Stegmann, Thomas J.; Kordyum, Vitaliy A.; Slavchenko, Iryna Yu.; Chernykh, Svitlana I.; Vozianov, Oleksandr

F.
PATENT ASSIGNEE(S): Germany
SOURCE: U.S. Pat. Appl. Publ., 42 pp., Cont.-in-part of U.S. Ser. No. 929,945.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004115769	A1	20040617	US 2003-649480	20030827
US 2002122792	A1	20020905	US 1999-358780	19990722
US 2002155532	A1	20021024	US 2001-929945	20010815
US 6642026	B2	20031104		
US 2003054492	A1	20030320	US 2002-280864	20021024

PRIORITY APPLN. INFO.: US 1998-93962P P 19980724
US 1999-358780 A2 19990722
US 2000-225406P P 20000815
US 2001-929945 A2 20010815

AB The present invention relates to the treatment of coronary heart disease by revascularization therapy, and more particularly to the intramyocardial injection of a pharmaceutical compn. comprising a recombinant fibroblast growth factor-1 protein or a fragment of a recombinant fibroblast growth factor-1 protein, optionally, with a physiol. glue for inducing local neoangiogenesis in ischemic myocardium. The invention also discloses methods of producing the recombinant fibroblast growth factor 1 protein and fragments. The methods involve phage-dependent delayed lysis of an Escherichia coli host cell for high-level prodn of sol., recombinant protein. The effects of the human aFGF 154, 146, and 140 recombinant proteins on angiogenesis were compared to pure brain-derived aFGF using the model of new blood vessel formation in chicken embryo chorio-allantoic membrane. Induced neoangiogenesis was also found in the ischemic rat heart model. Recombinant FGF -1 was used clin. in combination with coronary artery bypass graft in patients with coronary heart disease and FGF-1140 was used as sole therapy in 20 patients with coronary heart disease.

L12 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:674561 CAPLUS
DOCUMENT NUMBER: 137:196045
TITLE: Induction of neoangiogenesis in ischemic myocardium using a pharmaceutical composition containing FGF-1 and a physiological glue
INVENTOR(S): Stegmann, Thomas J.

PATENT ASSIGNEE(S): Germany
SOURCE: U.S. Pat. Appl. Publ., 16 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002122792	A1	20020905	US 1999-358780	19990722
US 2004115769	A1	20040617	US 2003-649480	20030827

PRIORITY APPLN. INFO.: US 1998-93962P P 19980724
US 1999-358780 A2 19990722
US 2000-225406P P 20000815
US 2001-929945 A2 20010815

AB The present invention relates to the treatment of coronary heart disease by revascularization therapy, and more particularly to the intramyocardial injection of a pharmaceutical compn. comprising fibroblast growth factor-1 and a physiol. glue for inducing local neoangiogenesis in ischemic myocardium.

L12 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:798674 CAPLUS
DOCUMENT NUMBER: 130:119673
TITLE: FGF-1: a human growth factor in the induction of neoangiogenesis
AUTHOR(S): Stegmann, Thomas J.
CORPORATE SOURCE: Department of Thoracic and Cardiovascular Surgery, Fulda Medical Center, Fulda, Germany
SOURCE: Expert Opinion on Investigational Drugs (1998), 7(12), 2011-2015
CODEN: EOIDER; ISSN: 1354-3784
PUBLISHER: Ashley Publications
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 25 refs. Currently available approaches for treating human coronary heart disease aim to relieve symptoms and the risk of myocardial infarction either by reducing myocardial oxygen demand, preventing further disease progression, restoring coronary blood flow pharmacol. or mech., or bypassing the stenotic lesions and obstructed coronary artery segments. Gene therapy, esp. using angiogenic growth factors, has emerged recently as a potential new treatment for cardiovascular disease. Following extensive exptl. research on angiogenic growth factors, the first clin. studies on patients with coronary heart disease and peripheral vascular lesions have been performed. The polypeptides fibroblast growth factor (

FGF) and vascular endothelial growth factor (VEGF) appear to be particularly effective in initiating neovascularization (neo-angiogenesis) in hypoxic or ischemic tissues. The first clin. study on patients with coronary heart disease treated by local intramyocardial injection of FGF-1 showed a 3-fold increase of capillary d. mediated by the growth factor. Angiogenic therapy of the human myocardium introduces a new modality of treatment for coronary heart disease in terms of regulation of blood vessel growth. Beyond drug therapy, angioplasty and bypass surgery, this new approach may evolve into a fourth principle of treatment of atherosclerotic cardiovascular disease.

REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1998:153567 CAPLUS

DOCUMENT NUMBER: 128:239666

TITLE: Induction of neoangiogenesis in ischemic myocardium by human growth factors: first clinical results of a new treatment of coronary heart disease

AUTHOR(S): Schumacher, B.; Pecher, P.; Von Specht, B. U.; Stegmann, Th.

CORPORATE SOURCE: Klinik fur Thorax-, Herz und Gefasschirurgie, Klinikum Fulda, Fulda, D-36043, Germany

SOURCE: Circulation (1998), 97(7), 645-650
CODEN: CIRCAZ; ISSN: 0009-7322

PUBLISHER: Williams & Wilkins

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The present article is a report of our animal expts. and also of the first clin. results of a new treatment for coronary heart disease using the human growth factor FGF-I (basic fibroblast growth factor) to induce neoangiogenesis in the ischemic myocardium.
FGF-I was obtained from strains of Escherichia coli by genetic engineering, then isolated and highly purified. Several series of animal expts. demonstrated the apathogenic action and neoangiogenic potency of this factor. After successful conclusion of the animal expts., it was used clin. for the first time. FGF-I (0.01 mg/kg body wt.) was injected close to the vessels after the completion of internal mammary artery (IMA)/left anterior descending coronary artery (LAD) anastomosis in 20 patients with three-vessel coronary disease. All the patients had addnl. peripheral stenoses of the LAD or one of its diagonal branches. Twelve weeks later, the IMA bypasses were selectively imaged by intra-arterial digital subtraction angiog. and quant. evaluated. In all the animal expts., the development of new vessels in the ischemic

myocardium could be demonstrated angiog. The formation of capillaries could also be demonstrated in humans and was found in all cases around the site of injection. A capillary network sprouting from the proximal part of the coronary artery could be shown to have bypassed the stenoses and rejoined the distal parts of the vessel. We believe that the use of FGF-I for myocardial revascularization is in principle a new concept and that it may be particularly suitable for patients with addnl. peripheral stenoses that cannot be revascularized surgically.

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 5 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:14963 BIOSIS

DOCUMENT NUMBER: PREV200100014963

TITLE: Neoangiogenesis by local gene therapy: A new therapeutic approach in the treatment of coronary artery disease.
Original Title: Neoangiogenese durch lokale Gentherapie: Ein neues therapeutisches Konzept in der Behandlung der koronaren Herzkrankheit.

AUTHOR(S): Schumacher, B. [Reprint author]; Hannekum, A.; Pecher, P.

CORPORATE SOURCE: Abt. Chirurgie IV, Herzchirurgie, Universitaetsklinik Ulm, Steinhoevelstr. 9, D-89075, Ulm, Germany

SOURCE: Zeitschrift fuer Kardiologie, (2000) Vol. 89, No. Supplement 7, pp. VII.23-VII.30. print.
CODEN: ZKRDAX. ISSN: 0300-5860.

DOCUMENT TYPE: Article

LANGUAGE: German

ENTRY DATE: Entered STN: 27 Dec 2000
Last Updated on STN: 27 Dec 2000

AB This article presents the results of our initial clinical experience with the human growth factor FGF-1 as applied to the ischemic human myocardium. After the completion of extensive preliminary animal experiments, the human angiogenetic growth factor FGF-1, obtained from a genetically transformed strain of E. coli was introduced into aortocoronary bypass surgery as an additional therapeutic agent. A double-blind study was carried out in a total of 40 patients with coronary heart disease. The patients were randomized into growth-factor and control groups, each containing 20 patients. All patients unterwent aortocoronary bypass surgery because of their coronary multivessel disease, in each case with an IMA bypass for the LAD and single venous bypasses for the RCX and/or RCA. In order to bridge over additional stenosis of the LAD or one of its branches, the human growth factor

FGF-1 was injected into the myocardium during the operation. In the control group heat-denatured growth factor was substituted for FGF-1. After three months as well as three years postoperatively, the IMA bypasses were selectively depicted by intraarterial DSA. These angiographies were then guantitatively evaluated. After the application of the growth factor, the formation of new vessels could be demonstrated after three months as well as three years postoperatively. A capillary network initiating from the coronary artery could be found and the computer-supported evaluation of the angiographs revealed a significant increase in the blood supply of the region of the myocardium injected. According to the angiographic findings there was also a clinical improvement of the patients with FGF-1 application compared to the patients of control group, concerning the NYHA classification as well as the need for anti-angina drug therapy. In the meantime, the results of other research groups support the evidence that the induction of neoangiogenesis by human growth factor could become a therapeutic approach especially in patients with diffuse coronary artery disease. Nevertheless further studies have to be carried out in order to prove the long-term clinical profit of those patients after the growth factor treatment.

L12 ANSWER 6 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2000:517402 BIOSIS
DOCUMENT NUMBER: PREV200000517402
TITLE: Induction of myocardial neoangiogenesis by human growth factors. A new therapeutic option in coronary heart disease.
AUTHOR(S): Stegmann, Thomas J. [Reprint author]; Hoppert, Thomas; Schneider, Andre; Gemeinhardt, Stefan; Koecher, Michael; Ibing, Rainer; Strupp, Gerhard
CORPORATE SOURCE: Klinik fuer Thorax-, Herz- und Gefaesschirurgie, Klinikum Fulda, Pacelliallee 4, D-36043, Fulda, Germany
SOURCE: Herz, (September, 2000) Vol. 25, No. 6, pp. 589-599. print. ISSN: 0340-9937.
DOCUMENT TYPE: Article
LANGUAGE: German
ENTRY DATE: Entered STN: 29 Nov 2000
Last Updated on STN: 11 Jan 2002
AB Currently available approaches for treating human coronary heart disease aim to relieve symptoms and the risk of myocardial infarction either by reducing myocardial oxygen demand, preventing further disease progression, restoring coronary blood flow pharmacologically or mechanically, or bypassing the stenotic lesions and obstructed coronary artery segments. Gene therapy, especially using angiogenic growth factors, has emerged recently as a potential new treatment for cardiovascular disease.

Following extensive experimental research on angiogenic growth factors, the first clinical studies on patients with coronary heart disease and peripheral vascular lesions have been performed. The polypeptides fibroblast growth factor (FGF) and vascular endothelial growth factor (VEGF) appear to be particularly effective in initiating neovascularization (neoangiogenesis) in hypoxic or ischemic tissues. The first clinical study on patients with coronary heart disease treated by local intramyocardial injection of FGF-1 showed a 3-fold increase of capillary density mediated by the growth factor. Also, angiogenic growth factor injection intramyocardially as sole therapy for end-stage coronary disease showed an improvement of myocardial perfusion in the target areas as well as a reduction of symptoms and an increase in working capacity. Angiogenic therapy of the human myocardium introduces a new modality of treatment for coronary heart disease in terms of regulation of blood vessel growth. Beyond drug therapy, angioplasty and bypass surgery, this new approach may evolve into a fourth principle of treatment of atherosclerotic cardiovascular disease.

L12 ANSWER 7 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1999:138037 BIOSIS
DOCUMENT NUMBER: PREV199900138037
TITLE: The stimulation of neoangiogenesis in the ischemic human heart by the growth factor FGF: First clinical results.
AUTHOR(S): Schumacher, B. [Reprint author]; Stegmann, T.; Pecher, P.
CORPORATE SOURCE: Abteilung Herzchirurgie, Universitatsklin. Ulm, D-89075 Ulm, Germany
SOURCE: Journal of Cardiovascular Surgery, (Dec., 1998) Vol. 39, No. 6, pp. 783-789. print.
CODEN: JCVSA2. ISSN: 0021-9509.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 31 Mar 1999
Last Updated on STN: 31 Mar 1999
AB Background. This paper is a report of our clinical experience with the human growth factor FGF as applied to the ischemic human myocardium. Methods. After the completion of extensive preliminary animal experiments, the growth factor FGF, obtained from genetically manipulated E. coli bacteria and highly purified, was introduced into aortocoronary bypass surgery as an additional therapeutic agent. A double blind study was carried out on 40 patients with CHD, separated into 'growth factor' and control groups, each containing 20 members. All the patients were treated for threefold vascular disease, in each case with an IMA bypass for the LAD and single venous bypasses for the RCX and/or RCA. In order to bridge over additional peripheral

stenoses in the LAD or one of its branches, human growth factor FGF was injected into the myocardium of those in the growth factor group. Twelve weeks later, the IMA bypasses were selectively demonstrated by intraarterial DSA. These angiographs were then quantitatively evaluated. Results. In all patients of the growth factor group, the formation of new vessels could be demonstrated in the region where FGF had been administered, in a manner strictly reminiscent of our experimental results. A capillary net sprouting from the coronary artery and making further connection with this vessel could be demonstrated, and the computer-supported evaluation of the angiographs showed a significant increase in the blood supply of the region of the myocardium injected. Conclusions. It is therefore our opinion that employment of the human growth factor FGF represents a useful extension to bypass surgery, particularly for patients with an additional peripheral stenosis that cannot he operatively revascularized.

L12 ANSWER 8 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1998:492531 BIOSIS
DOCUMENT NUMBER: PREV199800492531
TITLE: The stimulation of neo-angiogenesis in the ischemic heart by the human growth factor FGF.
AUTHOR(S): Schumacher, B. [Reprint author]; Von Specht, B.-U.; Haberstroh, J.; Pecher, P.
CORPORATE SOURCE: Abt. Herzchir., Universitaetsklin. Ulm, Steinhoevelstr. 9, D-89075 Ulm, Germany
SOURCE: Journal of Cardiovascular Surgery, (Aug., 1998) Vol. 39, No. 4, pp. 445-453. print.
CODEN: JCVSA2. ISSN: 0021-9509:
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 18 Nov 1998
Last Updated on STN: 18 Nov 1998
AB Background: The present article deals with the conduct of our animal experiments with the human growth factor FGF (fibroblast growth factor) and the results obtained therefrom. Methods: In order to establish the angiogenetic potential of FGF, this factor was first obtained from a genetically transformed strain of E. Coli and then isolated and highly purified. Afterwards the growth factor FGF has been used in several in vitro- and in vivo experiments in order to prove its influence on neo-angiogenesis in ischemic tissue. Results: In cultures of endothelial cells from the human great saphenous vein it has been possible to stimulate growth successfully with FGF obtained in this way, and a further increase in its action was brought about by the addition of heparin. in tritium-thymidine assays, the endothelial cell stimulating action of FGF was confirmed. It could also be shown

angiographically that administering FGF to the ischemic myocardium of these animals initiates the development of new vessels and we could demonstrate that a myocardial capillary network sprouting directly from the coronary vessels themselves can establish an alternative blood flow. These results were confirmed histologically by the significantly greater capillary density which appeared following the use of the growth factor. Conclusion: By using the human growth factor FGF, we have been able for the first time to understand the physiological processes of angiogenesis as they come into play during wound healing or the development of collaterals following tissue ischemia, and to use this knowledge for the production of new vessels in the ischemic hearts of rats and rabbits. Decisive for the future use of the factor in human patients particularly for the treatment of coronary heart disease (CHD) are the results of experimental investigation designed to excluded the possibility of the growth factor initiating or stimulating neoplasia.

L12 ANSWER 9 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1998:131418 BIOSIS
DOCUMENT NUMBER: PREV199800131418
TITLE: Induction of neoangiogenesis in ischemic myocardium by human growth factors: First clinical results of a new treatment of coronary heart disease.
AUTHOR(S): Schumacher, B. [Reprint author]; Pecher, P.; Von Specht, B. U.; Stegmann, T.
CORPORATE SOURCE: Klinik fuer Thorax-, Herz und Gefasschirurgie, Klinikum Fulda, Pacelliallee 4, D-36043 Fulda, Germany
SOURCE: Circulation, (Feb. 24, 1998) Vol. 97, No. 7, pp. 645-650. print.
CODEN: CIRCAZ. ISSN: 0009-7322.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 20 Mar 1998
Last Updated on STN: 20 Mar 1998
AB Background-The present article is a report of our animal experiments and also of the first clinical results of a new treatment for coronary heart disease using the human growth factor FGF-I (basic fibroblast growth factor) to induce neoangiogenesis in the ischemic myocardium. Methods and Results-FGF-I was obtained from strains of Escherichia coli by genetic engineering, then isolated and highly purified. Several series of animal experiments demonstrated the apadiogenic action and neoangiogenic potency of this factor. After successful conclusion of the animal experiments, it was used clinically for the first time. FGF-I (0.01 mg/kg body weight) was injected close to the vessels after the completion of internal mammary artery

(IMA)/left anterior descending coronary artery (LAD) anastomosis in 20 patients with three-vessel coronary disease. AU the patients had additional peripheral stenoses of the LAD or one of its diagonal branches. Twelve weeks later, the IMA bypasses were selectively imaged by intra-arterial digital subtraction angiography and quantitatively evaluated. In all the animal experiments, the development of new vessels in the ischemic myocardium could be demonstrated angiographically. The formation of capillaries could also be demonstrated in humans and was found in all cases around the site of injection. A capillary network sprouting from the proximal part of the coronary artery could be shown to have bypassed the stenoses and rejoined the distal parts of the vessel. Conclusions-We believe that the use of FGF-I for myocardial revascularization is in principle a new concept and that it may be particularly suitable for patients with additional peripheral stenoses that cannot be revascularized surgically.

=> D L13 IBIB TI 1-35

L13 ANSWER 1 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2006:314382 CAPLUS
DOCUMENT NUMBER: 144:362841
TITLE: Adenoviral PR39 improves blood flow and myocardial function in a pig model of chronic myocardial ischemia by enhancing collateral formation
AUTHOR(S): Post, Mark J.; Sato, Kaori; Murakami, Masahiro; Bao, Jialin; Tirziu, Daniela; Pearlman, Justin D.; Simons, Michael
CORPORATE SOURCE: Angiogenesis Research Center and Section of Cardiology, Dartmouth Medical School, Lebanon, NH, USA
SOURCE: American Journal of Physiology (2006), 290(3, Pt. 2), R494-R500
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Adenoviral PR39 improves blood flow and myocardial function in a pig model of chronic myocardial ischemia by enhancing collateral formation
REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 2 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:702515 CAPLUS
DOCUMENT NUMBER: 143:279591
TITLE: Transendocardial and transepicardial intramyocardial

fibroblast growth factor-2 administration: Myocardial and tissue distribution
AUTHOR(S): Laham, Roger J.; Post, Mark; Rezaee, Mehrdad; Donnell-Fink, Laurel; Wykrzykowska, Joanna J.; Lee, Seung U.; Baim, Donald S.; Sellke, Frank W.
CORPORATE SOURCE: The Cardiovascular Angiogenesis Center, Cardiology and Cardiovascular Division, Beth Israel Deaconess Medical Center, Boston, MA, USA
SOURCE: Drug Metabolism and Disposition (2005), 33(8), 1101-1107
CODEN: DMDSAI; ISSN: 0090-9556
PUBLISHER: American Society for Pharmacology and Experimental Therapeutics
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Transendocardial and transepicardial intramyocardial fibroblast growth factor-2 administration: Myocardial and tissue distribution
REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 3 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:612334 CAPLUS
DOCUMENT NUMBER: 143:126786
TITLE: Treatment of coronary or peripheral ischemia with heparin compound in combination with expression vector encoding angiogenic growth factor
INVENTOR(S): Uzan, Andre; Caron, Alexis
PATENT ASSIGNEE(S): Centelion, Fr.; Aventis Pharma S. A.
SOURCE: PCT Int. Appl., 41 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005063807	A2	20050714	WO 2004-EP14910	20041229
WO 2005063807	A3	20050929		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: US 2003-532993P P 20031229
TI Treatment of coronary or peripheral ischemia with heparin compound in combination with expression vector encoding angiogenic growth factor

L13 ANSWER 4 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:521720 CAPLUS
DOCUMENT NUMBER: 143:20021
TITLE: Non-mitogenic FGF-2 protects against ischemia and/or reperfusion injury
INVENTOR(S): Kardami, Elissavet
PATENT ASSIGNEE(S): Can.
SOURCE: Can. Pat. Appl., 56 pp.
CODEN: CPXXEB
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CA 2390285	AA	20031211	CA 2002-2390285	20020611

PRIORITY APPLN. INFO.: CA 2002-2390285 20020611
TI Non-mitogenic FGF-2 protects against ischemia and/or reperfusion injury

L13 ANSWER 5 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:301485 CAPLUS
DOCUMENT NUMBER: 143:53871
TITLE: Adenoviral Gene Transfer of FGF-5 to Hibernating Myocardium Improves Function and Stimulates Myocytes to Hypertrophy and Reenter the Cell Cycle
AUTHOR(S): Suzuki, Gen; Lee, Te-Chung; Fallavollita, James A.; Canty, John M.
CORPORATE SOURCE: VA WNY Health Care System and the Departments of Medicine and Physiology and Biophysics and the Center for Research in Cardiovascular Medicine at the University at Buffalo, Buffalo, NY, USA
SOURCE: Circulation Research (2005), 96(7), 767-775
CODEN: CIRUAL; ISSN: 0009-7330
PUBLISHER: Lippincott Williams & Wilkins

DOCUMENT TYPE: Journal
LANGUAGE: English
TI Adenoviral Gene Transfer of FGF-5 to Hibernating Myocardium Improves Function and Stimulates Myocytes to Hypertrophy and Reenter the Cell Cycle
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 6 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:1074120 CAPLUS
DOCUMENT NUMBER: 142:32967
TITLE: Plasmid encoding fibroblast growth factor for the treatment of hypercholesterolemia or diabetes associated angiogenic defects
INVENTOR(S): Caron, Alexis; Emmanuel, Florence; Caron, Anne; Finiels, Francoise; Michelet, Sandrine; Schwartz, Bertrand; Rouy, Didier; Branellec, Didier
PATENT ASSIGNEE(S): Gencell S.a.S., Fr.
SOURCE: PCT Int. Appl., 88 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004108167	A1	20041216	WO 2004-EP6903	20040604
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004244756	A1	20041216	AU 2004-244756	20040604
CA 2526792	AA	20041216	CA 2004-2526792	20040604
EP 1677831	A1	20060712	EP 2004-740314	20040604
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK				
US 2005096286	A1	20050505	US 2004-861906	20040607

PRIORITY APPLN. INFO.: US 2003-475959P P 20030605
US 2004-560915P P 20040409
US 2004-566193P P 20040428
WO 2004-EP6903 W 20040604

TI Plasmid encoding fibroblast growth factor for the treatment of hypercholesterolemia or diabetes associated angiogenic defects

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 7 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:493523 CAPLUS
DOCUMENT NUMBER: 141:47325
TITLE: Method of producing biologically active human acidic fibroblast growth factor and its use in promoting angiogenesis
INVENTOR(S): Stegmann, Thomas J.; Kordyum, Vitaliy A.; Slavchenko, Iryna Yu.; Chernykh, Svitlana I.; Vozianov, Oleksandr F.
PATENT ASSIGNEE(S): Germany
SOURCE: U.S. Pat. Appl. Publ., 42 pp., Cont.-in-part of U.S. Ser. No. 929,945.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004115769	A1	20040617	US 2003-649480	20030827
US 2002122792	A1	20020905	US 1999-358780	19990722
US 2002155532	A1	20021024	US 2001-929945	20010815
US 6642026	B2	20031104		
US 2003054492	A1	20030320	US 2002-280864	20021024

PRIORITY APPLN. INFO.: US 1998-93962P P 19980724
US 1999-358780 A2 19990722
US 2000-225406P P 20000815
US 2001-929945 A2 20010815

TI Method of producing biologically active human acidic fibroblast growth factor and its use in promoting angiogenesis

L13 ANSWER 8 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:213092 CAPLUS
DOCUMENT NUMBER: 140:418210
TITLE: Non-angiogenic FGF-2 protects the

ischemic heart from injury, in the presence or absence of reperfusion
AUTHOR(S): Jiang, Zhi-Sheng; Srisakuldee, Wattamon; Soulet, Fabienne; Bouche, Gerard; Kardami, Elissavet
CORPORATE SOURCE: Institute of Cardiovascular Sciences, St. Boniface Research Centre, Department of Human Anatomy and Cell Science and Physiology, University of Manitoba, Winnipeg, MB, R2H 2A6, Can.
SOURCE: Cardiovascular Research (2004), 62(1), 154-166
CODEN: CVREAU; ISSN: 0008-6363
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Non-angiogenic FGF-2 protects the ischemic heart from injury, in the presence or absence of reperfusion
REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 9 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:895952 CAPLUS
DOCUMENT NUMBER: 139:374668
TITLE: A randomized, double-blind, placebo-controlled trial of Ad5FGF-4 gene therapy and its effect on myocardial perfusion in patients with stable angina
AUTHOR(S): Grines, Cindy L.; Watkins, Matthew W.; Mahmarian, John J.; Iskandrian, Ami E.; Rade, Jeffrey J.; Marrott, Pran; Pratt, Craig; Kleiman, Neal
CORPORATE SOURCE: Angiogenic GENe Therapy (AGENT-2) Study Group, Department of Medicine, Section of Cardiology, William Beaumont Hospital, Royal Oak, MI, USA
SOURCE: Journal of the American College of Cardiology (2003), 42(8), 1339-1347
CODEN: JACCDI; ISSN: 0735-1097
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
TI A randomized, double-blind, placebo-controlled trial of Ad5FGF-4 gene therapy and its effect on myocardial perfusion in patients with stable angina
REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 10 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:610064 CAPLUS

DOCUMENT NUMBER: 139:160389
TITLE: Techniques and compositions for treating cardiovascular disease by in vivo gene delivery of angiogenic peptides and proteins
INVENTOR(S): Hammond, H. Kirk; Dillmann, Wolfgang; Giordano, Frank J.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 51 pp., Cont.-in-part of U.S. Ser. No. 609,080, abandoned.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 12
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003148968	A1	20030807	US 2001-847936	20010503
US 5792453	A	19980811	US 1995-485472	19950607
US 6100242	A	20000808	US 1997-722271	19971229
US 6174871	B1	20010116	US 1998-132167	19980810
WO 9940945	A2	19990819	WO 1999-US2702	19990209
WO 9940945	A3	19990930		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9947541	A1	19991125	AU 1999-47541	19990910
WO 2001034208	A1	20010517	WO 2000-US30345	20001103
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
ZA 2002003303	A	20030526	ZA 2002-3303	20020425
WO 2002089856	A1	20021114	WO 2002-US13990	20020503
WO 2002089856	C1	20040401		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,				

CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002305346	A1	20021118	AU 2002-305346	20020503
US 2004132190	A1	20040708	US 2003-741907	20031219

PRIORITY APPLN. INFO.:
US 1995-396207 B2 19950228
US 1995-485472 A2 19950607
US 1997-852779 B1 19970506
US 1997-722271 A2 19971229
US 1998-21773 B2 19980211
US 1998-68102 B2 19980430
US 1998-132167 A1 19980810
WO 1999-US2702 A2 19990209
US 1999-435156 B2 19991105
US 2000-609080 B2 20000630
WO 2000-US30345 A2 20001103
US 1995-481122 B2 19950607
AU 1996-50287 A3 19960227
WO 1996-US2631 W 19960227
US 1996-660387 B1 19960607
US 1998-98174 B1 19980616
US 2000-664127 A3 20000918
US 2001-847936 A 20010503
WO 2002-US13990 W 20020503

TI Techniques and compositions for treating cardiovascular disease by in vivo gene delivery of angiogenic peptides and proteins

L13 ANSWER 11 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:674561 CAPLUS
DOCUMENT NUMBER: 137:196045
TITLE: Induction of neoangiogenesis in ischemic myocardium using a pharmaceutical composition containing FGF-1 and a physiological glue
INVENTOR(S): Stegmann, Thomas J.
PATENT ASSIGNEE(S): Germany
SOURCE: U.S. Pat. Appl. Publ., 16 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002122792	A1	20020905	US 1999-358780	19990722
US 2004115769	A1	20040617	US 2003-649480	20030827

PRIORITY APPLN. INFO.: US 1998-93962P P 19980724
US 1999-358780 A2 19990722
US 2000-225406P P 20000815
US 2001-929945 A2 20010815

TI Induction of neoangiogenesis in ischemic myocardium using a pharmaceutical composition containing FGF-1 and a physiological glue

L13 ANSWER 12 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:637539 CAPLUS
DOCUMENT NUMBER: 137:163807
TITLE: Localized myocardial injection method for treating ischemic myocardium
INVENTOR(S): Palasis, Maria
PATENT ASSIGNEE(S): Boston Scientific Corporation, USA
SOURCE: PCT Int. Appl., 29 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002064157	A2	20020822	WO 2002-US3118	20020123
WO 2002064157	C2	20030605		
WO 2002064157	A3	20030807		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2433936	AA	20020822	CA 2002-2433936	20020123
US 2002172663	A1	20021121	US 2002-57409	20020123
EP 1361896	A2	20031119	EP 2002-720895	20020123

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
JP 2005501802 T2 20050120 JP 2002-563950 20020123
PRIORITY APPLN. INFO.: US 2001-263468P P 20010123
WO 2002-US3118 W 20020123
TI Localized myocardial injection method for treating ischemic myocardium

L13 ANSWER 13 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:569099 CAPLUS
DOCUMENT NUMBER: 137:304894
TITLE: Improved efficacy in cardiomyocyte transplantation for myocardial infarction therapy through prevascularization induced by controlled release of basic fibroblast growth factor
AUTHOR(S): Yamamoto, Masaya; Sakakibara, Yutaka; Nishimura, Kazunobu; Komeda, Masashi; Tabata, Yasuhiko
CORPORATE SOURCE: Institute for Frontier Medical Sciences, Kyoto University, Japan
SOURCE: Ensho, Saisei (2002), 22(3), 187-193
CODEN: ENSHCC
PUBLISHER: Nippon Ensho-Saisei Igakkai
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
TI Improved efficacy in cardiomyocyte transplantation for myocardial infarction therapy through prevascularization induced by controlled release of basic fibroblast growth factor

L13 ANSWER 14 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:93182 CAPLUS
DOCUMENT NUMBER: 134:335902
TITLE: Therapeutic angiogenesis in cardiology using protein formulations
AUTHOR(S): Post, M. J.; Laham, R.; Sellke, F. W.; Simons, M.
CORPORATE SOURCE: Angiogenesis Research Center and Cardiothoracic Surgery Division (FWS), Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA
SOURCE: Cardiovascular Research (2001), 49(3), 522-531
CODEN: CVREAU; ISSN: 0008-6363
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
TI Therapeutic angiogenesis in cardiology using protein formulations
REFERENCE COUNT: 122 THERE ARE 122 CITED REFERENCES AVAILABLE FOR
THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE

FORMAT

L13 ANSWER 15 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:591380 CAPLUS
DOCUMENT NUMBER: 133:305956
TITLE: Basic fibroblast growth factor caused no change in collateral flow or infarct size of acutely-infarcted myocardium in rats
AUTHOR(S): Inagaki, Masahiko; Kimura, Akio; Miyataka, Masaru; Ishikawa, Kinji
CORPORATE SOURCE: The First Department of Internal Medicine, Kinki University School of Medicine, Osakasayama, 589-8511, Japan
SOURCE: Tohoku Journal of Experimental Medicine (2000), 191(2), 101-111
CODEN: TJEMAO; ISSN: 0040-8727
PUBLISHER: Tohoku University Medical Press
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Basic fibroblast growth factor caused no change in collateral flow or infarct size of acutely-infarcted myocardium in rats
REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 16 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:81371 CAPLUS
DOCUMENT NUMBER: 132:203377
TITLE: Intrapericardial delivery of fibroblast growth factor-2 induces neovascularization in a porcine model of chronic myocardial ischemia
AUTHOR(S): Laham, Roger J.; Rezaee, Mehrdad; Post, Mark; Novicki, Debborah; Sellke, Frank W.; Pearlman, Justin D.; Simons, Michael; Hung, David
CORPORATE SOURCE: Angiogenesis Research Center and Interventional Cardiology Section, Department of Medicine, Harvard Medical School and Beth Israel Deaconess Medical Center, Boston, MA, USA
SOURCE: Journal of Pharmacology and Experimental Therapeutics (2000), 292(2), 795-802
CODEN: JPETAB; ISSN: 0022-3565
PUBLISHER: American Society for Pharmacology and Experimental Therapeutics
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Intrapericardial delivery of fibroblast growth factor-2 induces

neovascularization in a porcine model of chronic myocardial ischemia
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 17 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:359425 CAPLUS
DOCUMENT NUMBER: 131:14116
TITLE: New approaches to coronary heart disease: induction of neovascularization by growth factors
AUTHOR(S): Stegmann, Thomas J.
CORPORATE SOURCE: Department of Thoracic and Cardiovascular Surgery, Fulda Medical Center, Fulda, Germany
SOURCE: BioDrugs (1999), 11(5), 301-308
CODEN: BIDRF4; ISSN: 1173-8804
PUBLISHER: Adis International Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
TI New approaches to coronary heart disease: induction of neovascularization by growth factors
REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 18 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:798674 CAPLUS
DOCUMENT NUMBER: 130:119673
TITLE: FGF-1: a human growth factor in the induction of neoangiogenesis
AUTHOR(S): Stegmann, Thomas J.
CORPORATE SOURCE: Department of Thoracic and Cardiovascular Surgery, Fulda Medical Center, Fulda, Germany
SOURCE: Expert Opinion on Investigational Drugs (1998), 7(12), 2011-2015
CODEN: EOIDER; ISSN: 1354-3784
PUBLISHER: Ashley Publications
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
TI FGF-1: a human growth factor in the induction of neoangiogenesis
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 19 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:153567 CAPLUS
DOCUMENT NUMBER: 128:239666

TITLE: Induction of neoangiogenesis in ischemic myocardium by human growth factors: first clinical results of a new treatment of coronary heart disease
AUTHOR(S): Schumacher, B.; Pecher, P.; Von Specht, B. U.; Stegmann, Th.
CORPORATE SOURCE: Klinik fur Thorax-, Herz und Gefasschirurgie, Klinikum Fulda, Fulda, D-36043, Germany
SOURCE: Circulation (1998), 97(7), 645-650
CODEN: CIRCAZ; ISSN: 0009-7322
PUBLISHER: Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Induction of neoangiogenesis in ischemic myocardium by human growth factors: first clinical results of a new treatment of coronary heart disease
REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 20 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1994:432321 CAPLUS
DOCUMENT NUMBER: 121:32321
TITLE: Longitudinal changes in myocardial basic fibroblast growth factor (FGF-2) activity following coronary artery ligation in the dog
AUTHOR(S): Cohen, Michael V.; Vernon, Jackie; Yaghdjian, Vicken; Hatcher, Victor B.
CORPORATE SOURCE: Dep. Med. Physiol., Univ. South Alabama, Mobile, AL, USA
SOURCE: Journal of Molecular and Cellular Cardiology (1994), 26(5), 683-90
CODEN: JMCDAY; ISSN: 0022-2828
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Longitudinal changes in myocardial basic fibroblast growth factor (FGF-2) activity following coronary artery ligation in the dog

L13 ANSWER 21 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1994:316708 CAPLUS
DOCUMENT NUMBER: 120:316708
TITLE: Basic fibroblast growth factor enhances myocardial collateral flow in a canine model
AUTHOR(S): Unger, Ellis F.; Banai, Shmuel; Shou, Matie; Lazarous, Daisy F.; Jaklitsch, Michael T.; Scheninowitz, Mickey; Correa, Rosaly; Klingbeil, Corine; Epstein, Stephen E.

CORPORATE SOURCE: Cardiol. Branch, Natl. Heart, Lung, Blood Inst., Bethesda, MD, 20892, USA
SOURCE: American Journal of Physiology (1994), 266(4, Pt. 2), H1588-H1595
CODEN: AJPHAP; ISSN: 0002-9513
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Basic fibroblast growth factor enhances myocardial collateral flow in a canine model

L13 ANSWER 22 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1992:626960 CAPLUS
DOCUMENT NUMBER: 117:226960
TITLE: Salvage of infarcted myocardium by angiogenic action of basic fibroblast growth factor
AUTHOR(S): Yanagisawa-Miwa, Atsuko; Uchida, Yasumi; Nakamura, Fumitaka; Tomaru, Takanobu; Kido, Hideaki; Kamijo, Takeshi; Sugimoto, Tsuneaki; Kaji, Kazuhiko; Utsuyama, Masanori; et al.
CORPORATE SOURCE: Fac. Med., Univ. Tokyo, Tokyo, 113, Japan
SOURCE: Science (Washington, DC, United States) (1992), 257(5075), 1401-3
CODEN: SCIEAS; ISSN: 0036-8075
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Salvage of infarcted myocardium by angiogenic action of basic fibroblast growth factor

L13 ANSWER 23 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2006:257756 BIOSIS
DOCUMENT NUMBER: PREV200600254448
TITLE: Adenoviral PR39 improves blood flow and myocardial function in a pig model of chronic myocardial ischemia by enhancing collateral formation.
AUTHOR(S): Post, Mark J. [Reprint Author]; Sato, Kaori; Murakami, Masahiro; Bao, Jialin; Tirziu, Daniela; Pearlman, Justin D.; Simons, Michael
CORPORATE SOURCE: Maastricht Univ, Dept Physiol, Cardiovasc Res Inst Maastricht, Universiteitssingel 50, NL-6229 ER Maastricht, Netherlands
m.post@fys.unimaas.nl
SOURCE: American Journal of Physiology - Regulatory Integrative and Comparative Physiology, (MAR 2006) Vol. 290, No. 3, pp. R494-R500.

ISSN: 0363-6119.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 3 May 2006
Last Updated on STN: 3 May 2006
TI Adenoviral PR39 improves blood flow and myocardial function in a pig model of chronic myocardial ischemia by enhancing collateral formation.

L13 ANSWER 24 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2005:525488 BIOSIS
DOCUMENT NUMBER: PREV200510315451
TITLE: Intramyocardial injection of fibroblast growth factor-1 for treatment of refractory angina pectoris: The initial US experience.
AUTHOR(S): Wagoner, Lynne E. [Reprint Author]; Snavely, Daniel D.; Conway, Ginger A.; Hauntz, Elizabeth A.; Merrill, Walter H.
CORPORATE SOURCE: Univ Cincinnati, Cincinnati, OH USA
SOURCE: Circulation, (OCT 26 2004) Vol. 110, No. 17, Suppl. S, pp. 395.
Meeting Info.: 77th Scientific Meeting of the American-Heart-Association. New Orleans, LA, USA. November 07 -10, 2004. Amer Heart Assoc.
CODEN: CIRCAZ. ISSN: 0009-7322.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Dec 2005
Last Updated on STN: 1 Dec 2005
TI Intramyocardial injection of fibroblast growth factor-1 for treatment of refractory angina pectoris: The initial US experience.

L13 ANSWER 25 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2005:260533 BIOSIS
DOCUMENT NUMBER: PREV200510044966
TITLE: Adenoviral gene transfer of FGF-5 to hibernating myocardium improves function and stimulates myocytes to hypertrophy and reenter the cell cycle.
AUTHOR(S): Suzuki, Gen; Lee, Te-Chung; Fallavollita, James A.; Canty, John M. Jr [Reprint Author]
CORPORATE SOURCE: Univ Buffalo, Dept Med, Div Cardiol, Biomed Res Bldg,Room
347,3435 Main St, Buffalo, NY 14214 USA

canty@buffalo.edu
SOURCE: Circulation Research, (APR 15 2005) Vol. 96, No. 7, pp. 767-775.
CODEN: CIRUAL. ISSN: 0009-7330.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Jul 2005
Last Updated on STN: 14 Jul 2005
TI Adenoviral gene transfer of FGF-5 to hibernating myocardium improves function and stimulates myocytes to hypertrophy and reenter the cell cycle.

L13 ANSWER 26 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2005:30520 BIOSIS
DOCUMENT NUMBER: PREV200500033062
TITLE: Angiogenesis: A new therapeutic horizon in patients with stable angina pectoris.
Original Title: La angiogenesis, nuevo horizonte terapeutico en pacientes con angina cronica estable..
AUTHOR(S): Mautner, Branco [Reprint Author]; Cabello, Mariana; Garrido, Sol
CORPORATE SOURCE: Fdn Favaloro, ICYCC, Av Belgrano 1746, RA-1093, Buenos Aires, DF, Argentina
SOURCE: Prensa Medica Argentina, (August 2004) Vol. 91, No. 6, pp. 444-446. print.
CODEN: PMARAU. ISSN: 0032-745X.
DOCUMENT TYPE: Article
LANGUAGE: Spanish
ENTRY DATE: Entered STN: 12 Jan 2005
Last Updated on STN: 12 Jan 2005
TI Angiogenesis: A new therapeutic horizon in patients with stable angina pectoris.
Original Title: La angiogenesis, nuevo horizonte terapeutico en pacientes con angina cronica estable..

L13 ANSWER 27 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:437731 BIOSIS
DOCUMENT NUMBER: PREV200400439112
TITLE: Non-angiogenic FGF-2 protects the ischemic heart from injury, in the presence or absence of reperfusion.
AUTHOR(S): Jiang, Zhi-sheng; Srisakuldee, Wattamon; Soulet, Fablenne;

Bouche, Gerard; Kardami, Elissavet [Reprint Author]
CORPORATE SOURCE: Inst Cardiovasc SciSt Boniface Res CtrDept Human Anat and Cell Sci and Physiol, Univ Manitoba, 351 Tache Ave, Winnipeg, MB, R2H 2A6, Canada
ekardami@sbrc.ca
SOURCE: Cardiovascular Research, (April 1 2004) Vol. 62, No. 1, pp. 154-166. print.
CODEN: CVREAU. ISSN: 0008-6363.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Nov 2004
Last Updated on STN: 17 Nov 2004
TI Non-angiogenic FGF-2 protects the ischemic heart from injury, in the presence or absence of reperfusion.

L13 ANSWER 28 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:547302 BIOSIS
DOCUMENT NUMBER: PREV200300549128
TITLE: A randomized, double-blind, placebo-controlled trial of Ad5FGF-4 gene therapy and its effect on myocardial perfusion in patients with stable angina.
AUTHOR(S): Grines, Cindy L. [Reprint Author]; Watkins, Matthew W.; Mahmarian, John J.; Iskandrian, Ami E.; Rade, Jeffrey J.; Marrott, Pran; Pratt, Craig; Kleiman, Neal
CORPORATE SOURCE: Division of Cardiology, William Beaumont Hospital, 3601 West 13 Mile Road, 3rd Floor Heart Center, Royal Oak, MI, 48073-6769, USA
cgrines@beaumonthospitals.com
SOURCE: Journal of the American College of Cardiology, (October 15 2003) Vol. 42, No. 8, pp. 1339-1347. print.
ISSN: 0735-1097 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 19 Nov 2003
Last Updated on STN: 19 Nov 2003
TI A randomized, double-blind, placebo-controlled trial of Ad5FGF-4 gene therapy and its effect on myocardial perfusion in patients with stable angina.

L13 ANSWER 29 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:417477 BIOSIS
DOCUMENT NUMBER: PREV200100417477

TITLE: Intrapericardial delivery of fibroblast growth factor-2 induces neovascularization in a porcine model of chronic myocardial ischemia.
AUTHOR(S): Laham, Roger J. [Reprint author]; Rezaee, Mehrdad; Post, Mark; Novicki, Debborah; Sellke, Frank W.; Pearlman, Justin D.; Simons, Michael [Reprint author]; Hung, David
CORPORATE SOURCE: Angiogenesis Research Center, Beth Israel Deaconess Medical Center, Harvard Medical School, 330 Brookline Ave., Boston, MA, 02215, USA
rlaham@caregroup.harvard.edu
SOURCE: Journal of Pharmacology and Experimental Therapeutics, (February, 2000) Vol. 292, No. 2, pp. 795-802. print.
CODEN: JPETAB. ISSN: 0022-3565.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 29 Aug 2001
Last Updated on STN: 22 Feb 2002
TI Intrapericardial delivery of fibroblast growth factor-2 induces neovascularization in a porcine model of chronic myocardial ischemia.

L13 ANSWER 30 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:172487 BIOSIS
DOCUMENT NUMBER: PREV200100172487
TITLE: Therapeutic angiogenesis in cardiology using protein formulations.
AUTHOR(S): Post, Mark J. [Reprint author]; Laham, Roger; Sellke, Frank W.; Simons, Michael
CORPORATE SOURCE: Angiogenesis Research Center, Beth Israel Deaconess Medical Center, 330 Brookline Ave, Room SL 423, Boston, MA, 02215, USA
mpost@caregroup.harvard.edu
SOURCE: Cardiovascular Research, (16 February, 2001) Vol. 49, No. 3, pp. 522-531. print.
CODEN: CVREAU. ISSN: 0008-6363.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Apr 2001
Last Updated on STN: 18 Feb 2002
TI Therapeutic angiogenesis in cardiology using protein formulations.

L13 ANSWER 31 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2000:517402 BIOSIS
DOCUMENT NUMBER: PREV200000517402
TITLE: Induction of myocardial neoangiogenesis by human growth factors. A new therapeutic option in coronary heart disease.
AUTHOR(S): Stegmann, Thomas J. [Reprint author]; Hoppert, Thomas; Schneider, Andre; Gemeinhardt, Stefan; Koecher, Michael; Ibing, Rainer; Strupp, Gerhard
CORPORATE SOURCE: Klinik fuer Thorax-, Herz- und Gefaesschirurgie, Klinikum Fulda, Pacelliallee 4, D-36043, Fulda, Germany
SOURCE: Herz, (September, 2000) Vol. 25, No. 6, pp. 589-599. print.
ISSN: 0340-9937.
DOCUMENT TYPE: Article
LANGUAGE: German
ENTRY DATE: Entered STN: 29 Nov 2000
Last Updated on STN: 11 Jan 2002
TI Induction of myocardial neoangiogenesis by human growth factors. A new therapeutic option in coronary heart disease.

L13 ANSWER 32 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 1998:176890 BIOSIS
DOCUMENT NUMBER: PREV199800176890
TITLE: Effect of basic fibroblast growth factor on angiogenesis in the infarcted porcine heart.
AUTHOR(S): Watanabe, E.; Smith, D. M.; Sun, J.; Smart, F. W.; Delcarpio, J. B.; Roberts, T. B.; Claycomb, C. H., Jr Van Meter,and W. C. [Reprint author]
CORPORATE SOURCE: Dep. Biochem. and Mol. Biol., La. State Univ. Med. Cent., 1901 Perdido St., New Orleans, LA 70112, USA
SOURCE: Basic Research in Cardiology, (Feb., 1998) Vol. 93, No. 1, pp. 30-37. print.
CODEN: BRCAB7. ISSN: 0300-8428.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 20 Apr 1998
Last Updated on STN: 20 Apr 1998
TI Effect of basic fibroblast growth factor on angiogenesis in the infarcted porcine heart.

L13 ANSWER 33 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on

STN
ACCESSION NUMBER: 1998:131418 BIOSIS
DOCUMENT NUMBER: PREV199800131418
TITLE: Induction of neoangiogenesis in ischemic myocardium by human growth factors: First clinical results of a new treatment of coronary heart disease.
AUTHOR(S): Schumacher, B. [Reprint author]; Pecher, P.; Von Specht, B. U.; Stegmann, T.
CORPORATE SOURCE: Klinik fuer Thorax-, Herz und Gefasschirurgie, Klinikum Fulda, Pacelliallee 4, D-36043 Fulda, Germany
SOURCE: Circulation, (Feb. 24, 1998) Vol. 97, No. 7, pp. 645-650. print.
CODEN: CIRCAZ. ISSN: 0009-7322.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 20 Mar 1998
Last Updated on STN: 20 Mar 1998
TI Induction of neoangiogenesis in ischemic myocardium by human growth factors: First clinical results of a new treatment of coronary heart disease.

L13 ANSWER 34 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 1994:355412 BIOSIS
DOCUMENT NUMBER: PREV199497368412
TITLE: Longitudinal changes in myocardial basic fibroblast growth factor (FGF-2) activity following coronary artery ligation in the dog.
AUTHOR(S): Cohen, Michael V. [Reprint author]; Vernon, Jackie; Yaghdjian, Vicken; Hatcher, Victor B.
CORPORATE SOURCE: Dep. Physiol., MSB 3050, Univ. South Ala. Coll. Med., Mobile, AL 36688, USA
SOURCE: Journal of Molecular and Cellular Cardiology, (1994) Vol. 26, No. 5, pp. 683-690.
CODEN: JMCDAY. ISSN: 0022-2828.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Aug 1994
Last Updated on STN: 23 Aug 1994
TI Longitudinal changes in myocardial basic fibroblast growth factor (FGF-2) activity following coronary artery ligation in the dog.

L13 ANSWER 35 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN

ACCESSION NUMBER: 1994:296478 BIOSIS
DOCUMENT NUMBER: PREV199497309478
TITLE: Basic fibroblast growth factor enhances myocardial collateral flow in a canine model.
AUTHOR(S): Unger, Ellis F. [Reprint author]; Banai, Shmuel; Shou, Matie; Lazarous, Daisy F.; Jaklitsch, Michael T.; Scheinowitz, Mickey; Correa, Rosaly; Klingbeil, Corine; Epstein, Stephen E.
CORPORATE SOURCE: Cardiol. Branch, NHLBI, NIH, Building 10, Room 7B15, Bethesda, MD 20892, USA
SOURCE: American Journal of Physiology, (1994) Vol. 266, No. 4 PART 2, pp. H1588-H1595.
CODEN: AJPHAP. ISSN: 0002-9513.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 13 Jul 1994
Last Updated on STN: 13 Jul 1994
TI Basic fibroblast growth factor enhances myocardial collateral flow in a canine model.

=> FIL STNGUIDE

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- Search numbers may not be continuous; all searches are represented.
- Click on query # to add to strategy

Search	Most Recent Queries	Time	Result
#18	Search **ischemic region injection and myocardial and revascularization and fgf** Limits: **Entrez Date to 1998/07/24**	16:48:09	0
#16	Search **ischemic and myocardial and revascularization and fgf** Limits: **Entrez Date to 1998/07/24**	16:47:46	2
#15	Search **ischemic and myacardial and revasculization and FGF** Limits: **Entrez Date to 1998/07/24**	16:45:13	0
#14	Search **ischemic and myacardial and revasculization** Limits: **Entrez Date to 1998/07/24**	16:45:05	0
#12	Search **"Seitelberger R"[Author]**	14:51:45	66
#10	Search **Fasol R 1994**	14:50:14	7
#8	Search **myocardium and neoangiogenesis and fgf**	11:35:38	11
#7	Search **myocardium and neoangeogenesis and FGF**	11:24:19	165
#6	Search **schumacher B and neoangeogenesis**	11:23:58	237
#2	Search **schumacher B 1998 and neoangeogenesis**	11:23:45	26
#1	Search **schumacher B 1998**	11:20:28	26

Clear History

Jul 17 2006 06:31:01